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ORIGIN OF THE PYRAMIDAL TRACT IN THE CAT*

By J. W. LANCE† and R. L. MANNING‡

From the Brain Research Unit, Departments of Surgery, Anatomy and Physiology, University of Sydney, Australia

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Woolsey & Chang (1947) introduced a new technique into the study of the origin of the pyramidal tract when they recorded the arrival of antidromic pyramidal activity, initiated from the medulla oblongata, at the surface of the cortex. The present investigation has largely employed this useful method which has been extended by the insertion of microelectrodes into the cell layers of the cortex. By this means the electrical activity of single cells or groups of cells may be recorded as the cell bodies are depolarized by impulses passing antidromically up their axons. Accurate localization of cells giving rise to the pyramidal tract is thus possible, and the conduction velocities of their respective axons may be estimated. Results obtained by this method have been checked by the electrical stimulation of small areas of cortex and recording the consequent orthodromic pyramidal activity in medulla or cord.

METHODS

A series of twenty-five adult cats were used for these experiments. The animals were anaesthetized by Dial (Ciba) 0·5 ml./kg intraperitoneally, an additional 0·2–0·4 ml. of pentobarbitone solution (gr. 1/ml., 'Sagatal', May and Baker) being administered if necessary. After tracheal cannulation, the pyramidal tract was exposed in the medulla or cord by one of the following methods.

(1) Ventral approach to the medulla, by removal of larynx, part of pharynx and oesophagus. The longi capitis muscles were then reflected and a window made in the basiocciput with a dental burr. This approach enabled stimulating or recording electrodes to be placed directly in the pyramid with the animal in the lateral position.

(2) Dorsal approach to the fourth ventricle by removal of part of the occipital bone and cerebellum. Electrodes could then be inserted into the pyramids through the medulla, the animal being in the anatomical position.

(3) Laminectomy, exposing the spinal cord at cervical or lumbar segments, so that electrodes could be inserted into the lateral corticospinal tract.

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† National Health and Medical Research Council Fellow.

‡ Liston Wilson Fellow, University of Sydney.

The method chosen depended on the aspect of the cortex to be examined in the experiment and whether the animal was to be in the lateral or anatomical position.

In experiments on the medial surface of the brain, part of the opposite hemisphere was removed by diathermy and suction, and a section of the falx cerebri cut away.

After exposure of the appropriate area of cortex, skin flaps were elevated and the resulting 'dam' filled with paraffin oil which was maintained at 37° C for the duration of the experiment by a heating coil in a U-shaped glass tube, which was immersed in the paraffin. The rectal temperature of the animal was controlled at 38–39° C by means of a heating pad.

Recording microelectrodes were of steel, tapered electrolytically to less than 10 μ and insulated except at the tip. Stimulating electrodes were bipolar, the poles being steel electrodes as above, and aligned parallel 1 mm apart.

For antidromic studies the pyramidal tract was stimulated in the medulla. In cases when the stimulating electrodes were inserted through the floor of the fourth ventricle, the position of the electrodes was checked by recording from the surface of the cord at C_2 segment. A characteristic sequence of waves is recorded from the dorsum of the cord as the stimulating electrodes are inserted through the medulla (Lance, 1954) and at a depth of approximately 5 mm from the floor of the fourth ventricle, the double wave of the pyramidal complex is elicited. When the tips of the stimulating electrodes had thus been placed in position, the recording electrode was transferred to the cortex and orientated in a micromanipulator, cortical points being examined at intervals of 1 mm. The stimulating pulse, applied to the pyramid through a shielded transformer, was 50–70 V in amplitude, 50 μsec in duration.

In orthodromic studies the stimulating bipole was applied to the cortex at points separated by 2 mm, and the stimulus required was greater (115–160 V, 50 μsec pulse); the recording electrode was placed in the pyramidal tract in medulla or cord.

The amplified responses were displayed oscillographically and photographed on to high-speed emulsion plates.

At the conclusion of an experiment, the brain (and part of cord when necessary) was removed and after fixation in formalin, the surface of the brain was drawn or photographed and conduction distances carefully measured. The conduction distance from cerebral peduncle to cortical surface was estimated by the insertion of a threaded needle through the brain substance in a direct line. The distance thus obtained may be some millimetres shorter than the actual pathway of the axons. This is partially compensated, as electrodes recording cell activity were inserted 1·5–2 mm deep to the surface. Later the records obtained were projected from a photographic enlarger and traced on to the cortical map.

RESULTS

It is convenient to describe first the results of orthodromic stimulation as this is the less sensitive method of localization.

Form of orthodromic responses

Small areas of cortex were stimulated by means of bipolar electrodes, and the resulting activity in the pyramidal tract recorded from a microelectrode inserted into the ipsilateral tract in the medulla, or the contralateral cortico-spinal tract in the cord. The wave recorded was positive in sign owing to injury block in fibres surrounding the tip of the recording microelectrode. Each trough of such a wave represents an active group of fibres, and the onset of the initial positive deflexion precedes the arrival of the first impulses by about 0·1 msec (Lance, 1954).

The orthodromic wave in the pyramidal tract of medulla or cord elicited by stimulation of the pericruciate cortex showed conduction velocities ranging

from 4–9 m/sec (mean 7 m/sec) to 62–77 m/sec (mean 70 m/sec). Three discrete positive waves were recorded from the tract in the medulla and at the second cervical segment (Fig. 1*a*, *c*). This was an unexpected finding as the pyramidal tract in medulla and cord shows only two groups of activity (Bishop, Jeremy & Lance, 1953; Lance, 1954). To determine whether any one of these three waves

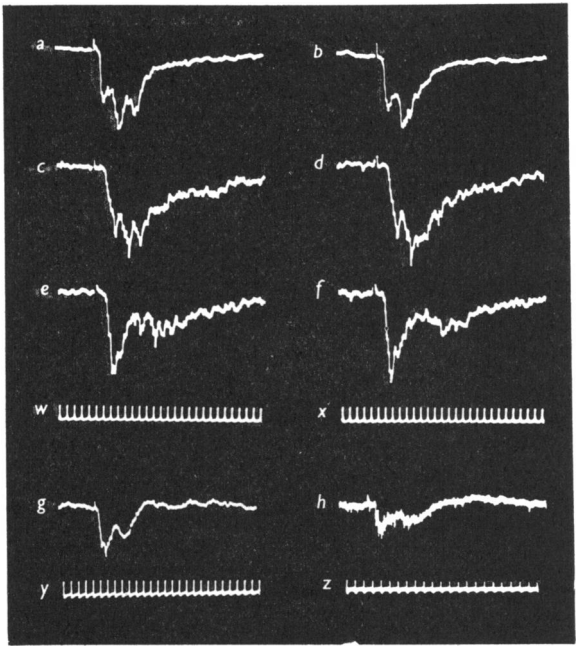

Fig. 1. Orthodromic records. *a*, from the pyramids of the medulla following stimulation of the pericruciate cortex (C.D. (conduction distance) 46 mm). *b*, as in *a*, preceded by a tetanus. The third wave of activity has ceased. *c*, from the contralateral corticospinal tract at C_2 segment on stimulation of the pericruciate cortex (C.D. 69 mm). *d*, as in *c* preceded by a tetanus. *e*, from the contralateral corticospinal tract at C_5 segment on stimulation of the pyramid in the medulla (C.D. 64 mm). *f*, as in *e* preceded by a tetanus. The second wave of the pyramid-cord response is seen to be of slower conduction velocity than the second wave of the cortex-cord response (*d*). Responses *c–f* are from the same preparation. Discussion in text. *g*, from the contralateral corticospinal tract at C_4 segment on stimulation of the pericruciate cortex (C.D. 85 mm). *h*, from the tract at L_1 segment on stimulation of the pericruciate cortex (C.D. 252 mm). In both *g* and *h* two waves are seen with time relations similar to those of the pyramid-cord response (*e*, *f*). Discussion in text. Time markers *w*, *x* 0·5 msec; *y*, 2·0 msec; *z*, 5·0 msec.

was initiated by a synaptic relay instead of by direct stimulation of pyramidal neurones, a tetanus of six impulses at 250/sec was applied to the preparation and, 9 msec after the last impulse of the tetanus, a test shock. Under these circumstances synapses cease to transmit impulses (Bishop & McLeod, to be published) and after such a tetanus the third wave of the complex disappeared (Fig. 1*b*, d), indicating that it was dependent upon a synaptic relay. The two

remaining waves reached voltage maxima at points corresponding to conduction velocities of 48–57 m/sec (mean 54 m/sec) and 22–25 m/sec (mean 24 m/sec) when recorded in the medulla, and the response returned to the base-line at a time corresponding to conduction at about 8 m/sec without any indication of a group slower than 24 m/sec (Fig. 1*b*). A similar post-tetanic record was obtained from the second cervical segment (Fig. 1*d*) with maxima showing average conduction velocities of 47 and 27 m/sec, while the return to the base-line suggested that more fibres were conducting in the range 7–18 m/sec, although no distinct slow group was seen. In records obtained from C_5 and L_1 segments on cortical stimulation, a slow group was obvious (Fig. 1*g*, *h*), and was unaffected by a preceding tetanus. Thus as the conduction distance in the spinal cord was increased relative to the conduction distance in the brain, the slow group became more prominent.

If the pyramidal tract be stimulated in the medulla instead of the cortex, two waves are recorded in the cord with peak amplitudes showing average conduction velocities of 50 and 14 m/sec (Lance, 1954). In Fig. 1, *e* and *f* are responses from the tract at C_5 segment, stimulation taking place in the medulla (conduction distance 64 mm), while *d* is the post-tetanic response from C_2 segment in the same animal, stimulation taking place in the cortex (conduction distance 69 mm). It can be seen that the second wave of the cortex-cord response reaches its maximum at a higher conduction velocity than the second wave of the medulla-cord response in the same preparation.

To summarize these findings, it appears that the range of velocities in all portions of the pyramidal tract averages 7–70 m/sec, and that the first pyramidal group is maximal at approximately 50 m/sec in all sections of the tract. However, the second group is maximal at 22–25 m/sec from cortex to brain-stem and at 11–16 m/sec from medulla to cord segments. This alteration in conduction velocity is considered to be due to reduction in size of some axons following the distribution of collaterals to pons and medulla.

Distribution of orthodromic responses

Pyramidal activity in the medulla is greatest when the region around the cruciate sulcus is stimulated but some activity can be elicited from a wide area of cortex (Fig. 2). No response occurs when the medial third of the anterior sigmoid gyrus is stimulated. This area was classified histologically as 'frontal' by Campbell (1905) and Langworthy (1928) and as 'area 6' by Winkler & Potter (1914). The lateral two-thirds of the anterior sigmoid gyrus and the whole posterior sigmoid gyrus, together with the coronal gyrus, constitute the 'motor area' of Campbell and 'area 4' of Winkler & Potter. This area gives rise to large orthodromic potentials with the time relations described above. The source of most fast activity appears to be at the lateral end of the cruciate sulcus. As the stimulating electrodes are moved medially along the posterior

sigmoid gyrus in 2 mm steps, fast conduction velocities (40–70 m/sec) become less prominent until at the medial end there is little activity faster than 40 m/sec. There is a similar gradient along the anterior sigmoid gyrus from lateral to medial until the junction of middle and medial thirds where area 4 merges into area 6, from which no orthodromic response can be derived.

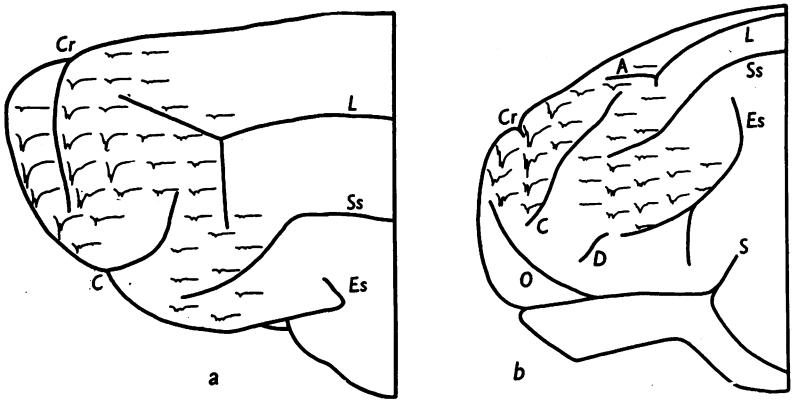

Fig. 2. Origin of the pyramidal tract in the cat determined by orthodromic studies. Dorsal and lateral aspects of the brain are shown. Records obtained from the contralateral tract at the second cervical segment have been drawn on the cortical map at the point from which they were elicited. The sulci (after Campbell) are: *A*, ansate; *C*, coronal; *Cr*, cruciate; *D*, diagonal; *Es*, anterior ectosylvian; *L*, lateral; *O*, orbital; *S*, sylvian; *Ss*, suprasylvian. Winkler & Potter (1914) term the diagonal and orbital sulci the orbital and praesylvian sulci respectively.

In the cat there is a dimple in the cortex lying between the cruciate and ansate sulci which appears to be the homologue of the primate central sulcus, for here the motor cortex gives place to typical sensory cortex (Campbell, 1905). At this point the form of the orthodromic potential also undergoes change, there being less fast activity posterior to the homologue of Rolando. Small potentials can be elicited from almost all of Campbell's 'sensory cortex' (subdivided somewhat indistinctly by Winkler & Potter into areas, 3, 1 and 5). The responses are usually larger in the inferior part of the 'sensory cortex' between the anterior limb of the suprasylvian sulcus and the diagonal sulcus, the region known as the 'secondary somatic area' (Garol, 1942*a*; Woolsey & Chang, 1947). These responses have the same time relations and grouping as those of the pericruciate area but are smaller in amplitude.

Form of antidromic responses

When the pyramids of the medulla are electrically stimulated, the waves of activity spreading antidromically along pyramidal axons may be recorded from the surface of the cortex (Woolsey & Chang, 1947). These surface

recordings are predominantly positive in sign (Fig. 3*a*), presumably because the pyramidal activity dies away in the cell layers of the cortex deep to the recording electrode on the surface. If a microelectrode be now inserted through the pia and racked gently into the underlying cortex, a positive-negative diphasic wave with a negative peak at a time corresponding with a conduction velocity of 50 m/sec becomes apparent (Fig. 4*a*), indicating that the micro-

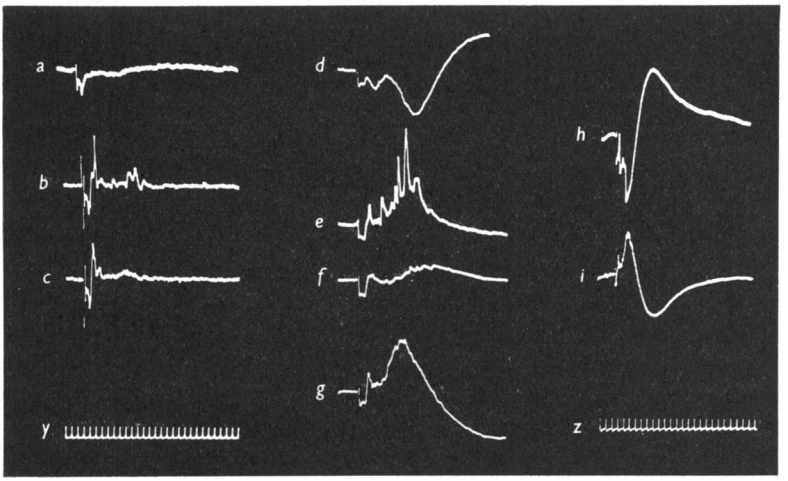

Fig. 3. Antidromic records. *a*, from the cortical surface anterior to the cruciate sulcus, the pyramid being stimulated in the medulla (C.D. 49 mm). *b*, on insertion of the microelectrode into the cortex to a depth of approx. 1·5 mm at the same recording site as *a*. *c*, with the microelectrode inserted into the white matter, approx. 3·0 mm deep, same site as *a*. *d*, from the cortical surface posterior to the cruciate sulcus, the medial lemniscus being stimulated in the medulla (C.D. 46 mm). *e*, on insertion of a microelectrode beneath the pia to a depth of approx. 1·5 mm, same recording site as *d*. *f*, as in *e*, preceded by a tetanus. *g*, with the microelectrode inserted into the white matter, approx. 3·0 mm deep, same site as *d*. *h* and *i*, as *d* and *g* with a slower time sweep, showing that the lemniscus waves recorded deep to the cell layers are the mirror image of those recorded on the surface. Time markers: *y*, 0·5 msec (for *a–g*); *z*, 2·0 msec (for *h*, *i*).

electrode is approaching the region of activity; then, with a further slight advancement of the microelectrode sudden changes of potential in the form of spikes and waves appear in the record (Figs. 3*b*, 4*b*). These are linked to the stimulus, distinct from the random activity which occurs from time to time while the recordings are being made. These spikes are recorded from the cell layers of the cortex at a depth of approximately 1·2–2·0 mm. It is dificult to determine the depth exactly owing to pial dimpling, and iron deposition from the electrode tip is unsatisfactory in these experiments as movement of the cortex occurs with respiration, so that the position of the electrode tip may alter between recording and deposition of iron. The activity usually occurred

in two main groups in the pericruciate region (Fig. 3*b*). As the electrode is racked further, spikes are seen no longer and negative-going waves are recorded when the electrode tip is among axons in the white matter (Fig. 3*c*).

The only constant wave recorded from the white matter is a positive-negative wave with a peak at a conduction velocity of approximately 50 m/sec due to the electrical field generated by the arrival at the cortex of the first pyramidal

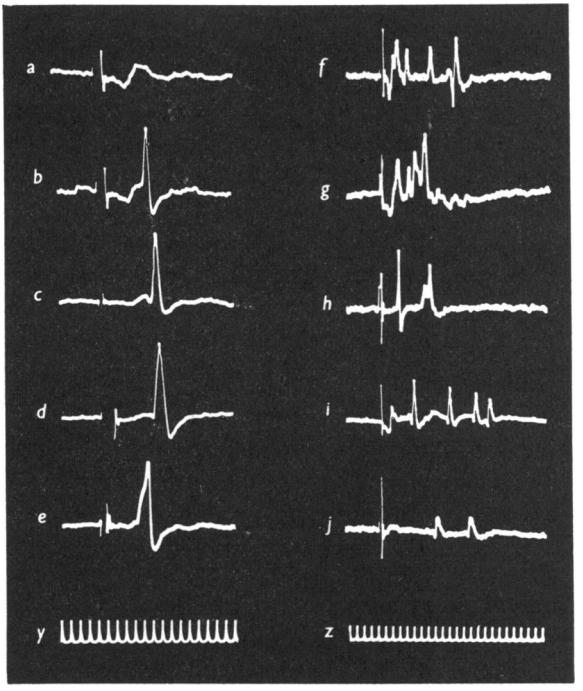

Fig. 4. Antidromic records. *a*, positive-negative diphasic wave recorded by a microelectrode deep to the pia in motor and sensory areas of cortex when the pyramid is adequately stimulated in the medulla (C.D. 51 mm). *b*, spike potential superimposed on this wave as the microelectrode is inserted further. *c–e*, typical spike potentials recorded from the cell layers of the cortex on stimulation of the pyramids in the medulla—from the posterior sigmoid gyrus (C.D. 46 mm), middle supra-sylvian gyrus (C.D. 44 mm), and secondary somatic area (C.D. 38 mm) respectively. *f, g*, typical rows of spikes recorded from pericruciate cortex (C.D. 48 mm). *h*, from the medial surface of the hemisphere, area 4 (C.D. 48 mm.) *i*, from the secondary somatic area (C.D. 37 mm). *j*, from the medial third of the anterior sigmoid gyrus, area 6 (C.D. 50 mm). Time markers: *y* 0·2 msec; *z* 0·5 msec.

group volley. It occasionally bears a second negative deflexion on the descending phase, and may be followed by a slow negative wave of low amplitude (Fig. 3*c*). It is difficult to exclude lemniscus fibres completely as a cause of this slow wave, for when the pyramid is stimulated in the medulla the medial lemniscus is liable to be activated as well. Fortunately, the full lemniscus response can be recognized easily. As Woolsey & Chang (1947) pointed out,

it takes the form of a slow positive-negative wave as recorded on the cortical surface (Fig. 3*d*, *h*). When a microelectrode is inserted into the cortex, spikes are recorded superimposed on a negative wave corresponding to the positive phase of the lemniscus surface wave (Fig. 3*e*) and are unlikely to be confused with the spike responses of antidromic pyramidal impulses which arise from a straight base-line (Fig. 4*f–j*). Following a tetanus (6 impulses at 250/sec) the lemniscus response largely, but not entirely, disappears (Fig. 3*f*). This residual negative wave which may be due to direct, non-synaptic, lemniscus fibres cannot be distinguished from late pyramidal activity. Deep to the cell layers of the cortex the lemniscus waves are a mirror image of those recorded on the surface (Fig. 4*g*, *i*).

The localization of the origin of the pyramidal tract by antidromic stimulation was therefore based on the distribution of spikes recorded in the immediate vicinity of cortical cells, rather than on the slow potential changes recorded superficial and deep to cell layers which are caused by widespread electrical fields and can so easily be confused by the activity of medial lemniscus.

Following stimulation of the pyramids in the medulla, a microelectrode inserted into the pericruciate cortex of area 4 recorded showers of spike potentials at all placements of the electrode (Fig. 4*f–h*). Away from area 4, only one or two spikes appeared, and no particular cortical point could be relied on to give a spike response, even though the diphasic wave of the electrical field (Fig. 4*a*) was usually recorded, and adjacent cortex 1 mm from the point gave rise to typical spikes. The map of responsive points (Fig. 5) is a composite of many experiments, but the points plotted in the areas other than pericruciate were not consistent from one experiment to another, although the ratio of positive to negative points indicated on the map is quite typical. It was frequently found that spike responses could no longer be obtained from any area after the experiment had been in progress about 3 hr. More spikes would probably have been recorded in all areas and the preparations lasted longer had the animals not been anaesthetized by barbiturates (Li & Jasper, 1953). On occasions a run of spikes occurred at fairly regular intervals which suggested that one or more cells were firing repetitively (Fig. 4*i*). This could be explained by recurrent pyramidal fibres synapsing with interneurones that return to activate cells at the recording site.

The form of an individual spike is a sharply rising negative wave followed by a positive wave smaller in amplitude (Fig. 4*b–d*). Single unit spikes of similar conformation have been reported occurring spontaneously in the cat cortex by Li & Jasper (1953), and following afferent stimulation by Amassian (1953). The latter found the duration of the initial negative spike usually to lie between 0·3 and 0·64 msec. On accasions we have seen the negative component of very short duration (0·2 msec) and at other times a compound spike composed of two or more units (Fig. 4*e*). From the rapidity with which spikes

appear and disappear with only minor adjustments of the electrode, it is probable that they may only be recorded when the tip is very close to the cell body; Li & Jasper estimated the potential field to be of the order of 60 μ in diameter. Spikes were photographed for record purposes only when they were

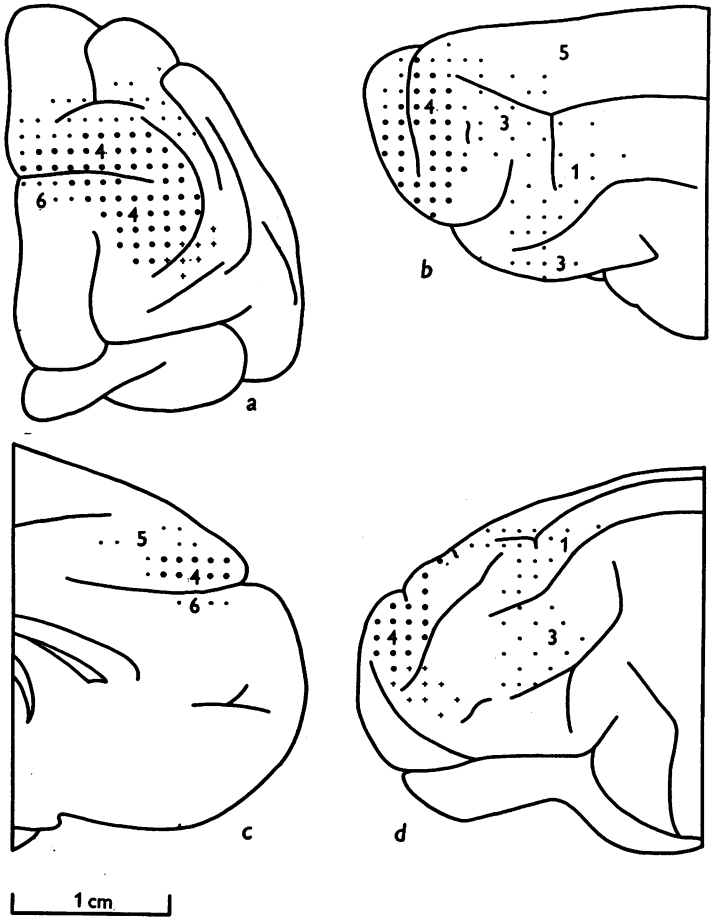

Fig. 5. Origin of the pyramidal tract in the cat determined by the distribution of spike potentials consequent on antidromic activation of the tract in the medulla. Aspects of the brain illustrated are *a*, anterior; *b*, dorsal; *c*, medial; *d*, lateral. The large dots indicate consistent responses composed of many spikes, the small dots indicate variable responses usually of one or two spikes, and the crosses occasional responses attributed to the stimulation of cortico-quintine fibres. The areas have been given numbers after Winkler & Potter (1914) and Garol (1942*b*).

consistently present for a number of time-base sweeps, and obviously linked to the stimulus, to avoid confusion with random activity. The spike is almost certainly due to depolarization of a cell body following the arrival of an anti-dromically conducted impulse in its axon.

Distribution of antidromic responses

Points yielding a spike response are illustrated in the cortical maps of Fig. 5. Conduction time for each spike was measured to the onset of the sharp negative upsweep.

The frontal cortex of the cat ('area 6' of Winkler & Potter, 1914) comprises the medial third of the anterior sigmoid gyrus and continues on to the medial surface of the hemisphere inferior to the cruciate sulcus. Although no response was recorded in the pyramids when this area was stimulated in orthodromic experiments, indicating the paucity of fibres which originate from it, some spikes were always recorded from this area in antidromic experiments (Fig. 4*j*). They were few in number, with a latency corresponding to a slow conduction velocity (7–14 m/sec) and therefore presumably from axons of fine calibre of the order of 1μ axon diameter. However the possibility that they were recorded from cells stimulated by recurrent pyramidal collaterals could not be excluded.

Within the 'motor area' or 'area 4' showers of spikes were recorded consistently with each placement of the electrode. These frequently appeared in two groups both at the lateral end of the cruciate sulcus and anterior to it, but elsewhere there was usually a continuous row of spikes (Fig. 4*f*, *g*) covering a range of conduction velocities 5–69 m/sec. In a few experiments no activity was seen faster than 50 m/sec. Closer to the mid-line in the posterior sigmoid gyrus there were few fast spikes (consistent with the orthodromic experiments), the fastest being about 40 m/sec. Similarly, as points in the anterior sigmoid gyrus were plotted medially, the conduction velocity of the fastest activity dropped to about 30 m/sec; then, as area 6 was reached, to 12–14 m/sec. In the coronal gyrus, between the cruciate and coronal sulci, responses occurred in the range 7–60 m/sec, there usually being one or two unresponsive points next to the coronal sulcus. Inferolateral to the coronal sulcus, an area shown previously to be connected with facial movement (Ward & Clark, 1935; Garol, 1942*a*), results were inconsistent. In some experiments responses were recorded (7–30 m/sec with an occasional spike at 60 m/sec), but in other experiments no responses were obtained from this region. These spikes when present are considered to follow the stimulation of corticoquintine fibres in the medulla and their variability therefore to be due to the numbers of such fibres present at the level of the pyramid stimulated in a given experiment. Area 4 continues on the medial surface of the hemisphere superior to the cruciate sulcus and responses are recorded here of 8–45 m/sec (Fig. 4*h*).

Posterior to the homologue of Rolando where motor cortex gives way to sensory, responses became less frequent, and more consisted of single spikes. Although orthodromic stimulation suggested that there were few fibres in the sensory cortex with conduction velocities greater than 40 m/sec, antidromic

spikes were recorded over the whole range up to 58 m/sec. Similar responses were obtained throughout Campbell's 'sensory cortex'. A few spikes of slow velocity (8–20 m/sec) were obtained from the parietal cortex on the medial surface of the hemisphere. The responses from the anterior part of the lateral gyrus and the anterior suprasylvian gyrus were similar to those immediately posterior to the homologue of Rolando. The area inferior to the anterior limb of the suprasylvian sulcus, the 'secondary somatic area', showed little difference in the number of spike responses from the remainder of the parietal cortex. The distribution of conduction velocities was the same as in the pericruciate area and some fast spikes of 60–65 m/sec were recorded.

The relative number of fibres arising from different cortical areas can be roughly estimated by counting the spikes recorded from these areas in all experiments. As points 1 mm apart were examined in all areas, this may be regarded as a fair method of sampling but it ignores a lot of the buried cortex deep in the cruciate and other sulci. It appears probable that at least two-thirds of pyramidal fibres arise anterior to the homologue of Rolando. The remaining one-third, or less, arise from parietal areas. Approximately 3% of the total fibres have conduction velocities greater than 60 m/sec; most of these occur in the pericruciate area with an occasional one in the 'secondary somatic area'.

DISCUSSION

Consideration of all experiments, antidromic and orthodromic, shows that the characteristic range of velocities in the pyramidal tract is 7–70 m/sec, values that agree well with those previously determined for the tract in the spinal cord (Lance, 1954). The orthodromic response recorded in the pyramids or in the upper cervical segments demonstrates three waves of activity, the first two of which are undisturbed by a tetanus, and are apparently caused by the activity of two pyramidal fibre groups. The third wave depends on a synaptic relay. Patton & Amassian (1952) reported only one direct wave, and a number of indirect or relayed waves in the corticospinal response, presenting evidence that this relay occurs in the cortex. Wall, Rémond & Dobson (1953), while demonstrating the effect of visual stimulation in facilitating pyramidal activity, found three orthodromic pyramidal waves similar to those reported here, but followed Patton & Amassian's interpretation, considering that only the first of these waves was caused by unrelayed fibres. As two waves have survived tetanic stimulation in all our orthodromic experiments and the third wave has consistently succumbed, there is good evidence that the direct pyramidal response represents two fibre groups. The waves of activity of these groups attain their greatest amplitude at 48–57 and 22–25 m/sec respectively in their path from cortex to brain stem. When the pyramidal tract is stimulated in the medulla, two groups can be recorded in the contralateral corticospinal tract down to lumbar segments of the cord, reaching maxima at 45–56 and

11–16 m/sec respectively (Lance, 1954). There is thus little alteration in the conduction velocity of the first group in its path from cortex to cord, but the second group alters markedly from approximately 24 m/sec to approximately 14 m/sec. This is probably related to the large numbers of collaterals distributed by the pyramidal tract to pontine and medullary nuclei. Brookhart & Morris (1948) examined quantitatively the histology of the pyramids in the cat medulla and found no evidence of segregation into fibre groups. However, Häggquist (1937) published a spectrum of fibre-diameters in the pyramid of *Macacus rhesus* which showed a large group of fibres 1–3 μ in diameter with the suggestion of a second mode at 6–7 μ diameter, and in 1942 Szentàgothai-Schimert reported two modes of distribution in the human pyramid at 1 and 7 μ respectively.

The pyramidal tract in the cat has been found to arise from the 'frontal', 'motor' and 'sensory' areas of Campbell (1905). It is possible that some of the spikes recorded from parietal and frontal areas could result from depolarization of cells by recurrent pyramidal collaterals. However, spikes have been recorded in parietal areas with latencies that preclude an interposed synapse, whereas this possibility cannot be eliminated in the frontal region. The distinction between 'frontal' and 'motor' cortex (areas 6 and 4) is clear histologically and occurs approximately at the junction of the medial third and lateral two-thirds of the anterior sigmoid gyrus (Campbell, 1905; Winkler & Potter, 1914; Langworthy, 1928). Evidence of a contribution from area 6 to the pyramidal tract in monkey and man is doubtful as there are conflicting reports in the literature (Mettler, 1935; Hoff, 1935; Kennard, 1935; Levin, 1936; Verhaart & Kennard, 1940; Minckler, Klemme & Minckler, 1944). Time relations of the few spikes recorded from the frontal region in the present study indicate that any fibres arising from this area are of slow conduction velocity, therefore fine in calibre and difficult to trace by any histological method, a difficulty that may account for the inconclusive findings in experimental work in the monkey.

It is now established that a large number of pyramidal fibres arise from parietal areas in the monkey (Levin & Bradford, 1938; Lassek, 1942; Peele, 1942), cat (Gobbel & Liles, 1945) and dog (Spiegel, Weston & Oppenheimer, 1943). These findings are confirmed by the electrophysiological studies of Woolsey & Chang (1947) and, for the cat, by the present study. The histology of Campbell's 'sensory area' has not been adequately studied in the cat but includes cortex with the cytoarchitectonic structure of areas 3, 1 and 5 (Winkler & Potter, 1914). Garol (1942*b*) has subdivided this area on the basis of strychnine neuronography and the origin of the pyramidal tract includes his areas 3, 2s, 1 and 5a.

At least two-thirds of pyramidal fibres take origin anterior to the homologue of Rolando in the cat, and the same ratio has been recently reported by Lassek

(1952) for the monkey. About 3% of all antidromic spikes recorded in this study indicated conduction velocities greater than 60 m/sec, and most of these occurred around the lateral end of the cruciate sulcus, although some were found in the 'secondary somatic area'. In 1878, Lewis described the largest 'Betz cells' in the cat as being in the former region, averaging in size $83 \times 37 \mu$. It seems reasonable to assume that these large cells give rise to axons with the fastest activity of 60–70 m/sec, which corresponds to an axon diameter of $7–8 \mu$ (Gasser & Grundfest, 1939).

Fibres of conduction velocity less than 60 m/sec were distributed evenly throughout all areas as judged by the frequency of spike discharges in antidromic studies. However, orthodromic stimulation suggested that the velocity of most fibres from cortex outside the pericruciate and secondary somatic areas was considerably slower than that of fibres from these two areas. The orthodromic method is probably more reliable here as many of the antidromic impulses in small axons may be blocked at the axon hillock and fail to discharge their parent cell. In summary, it may be stated that some fibres from both main pyramidal groups arise from parietal areas 3, 1 and 5, as well as from area 4.

The motor cortex of the cat has been subjected to many stimulation experiments since Ferrier (1890) showed in 1870 that hindlimb movements could be elicited from the posterior sigmoid gyrus and forelimb movements from the anterior sigmoid. The most recent complete study of this type is that of Garol (1942*a*), who found that the distal part of the hindlimb was served by the medial half of the posterior sigmoid gyrus, and hip movements by the lateral half. The anterior sigmoid and coronal gyri initiated forelimb movements. Movements were also obtained from the 'secondary somatic area'—facial movements from the superior part, and reciprocal movements of one forelimb and the opposite hindlimb from the inferior part. The areas found responsive to stimulation by Garol are those which are found in this study to make the greatest contribution to the pyramidal tract.

Our findings thus confirm and expand those of Woolsey & Chang (1947), the main point of difference being the range of conduction velocities in the tract which they estimated at 2–100 m/sec, an estimate based on recording of waves from the surface of the cortex. The onset and termination of these surface waves are difficult to define with accuracy, whereas spike potentials from the cell layers are clear-cut, and the range of velocities determined from them in this paper has been checked by orthodromic stimulation.

SUMMARY

1. The origin of the pyramidal tract in the cat has been studied by orthodromic and antidromic activation of tract fibres. The average range of conduction velocities in the tract was found to be 7–70 m/sec.

2. The pyramidal tract comprises two groups of fibres in brain and spinal cord. The wave of activity caused by the first of these groups reaches a maximum amplitude at a conduction velocity of approximately 50 m/sec throughout its path from cortex to lumbar spinal cord segments; the second reaches a maximum at approximately 24 m/sec from cortex to brain-stem, and approximately 14 m/sec from brain-stem to cord. This slowing of conduction velocity is attributed to diminution in axon diameter following distribution of collaterals to nuclei of the pons and medulla.

3. At least two-thirds of pyramidal fibres take origin anterior to the homologue of Rolando, the remaining one-third or less arising from 'sensory' cortex. About 3% of fibres have conduction velocities greater than 60 m/sec, and these arise mainly from the pericruciate cortex, a few arising from the 'secondary somatic area'.

4. Some fibres from both pyramidal groups arise from cortical areas 4, 3, 1 and 5, the average conduction velocity of parieto-spinal fibres being less than that of fibres from the two 'motor' areas. Area 6 may contribute a few fibres of slow conduction velocity (7–14 m/sec) to the tract.

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